

4. Tracey, J.K., Fong, Y., Hesse, D.G. *et al.* Anti-cachectin/ TNF monoclonal antibodies prevent septic shock during lethal bacteraemia. *Nature* 1987, **330**: 662–664.
5. Welsh, C.H., Lien, D., Worthen, G.S. & Weil, J.V. Pentoxifylline decreased endotoxin-induced pulmonary neutrophil sequestration and extravascular protein accumulation in the dog. *Am Rev Respir Dis* 1988, **138**: 1106–1114.
6. Schonharting, M.M. & Schade, U.F. The effect of pentoxifylline in septic shock-new pharmacologic aspects of an established drug. *J Med* 1989, **20**: 97–105.
7. Streiter, R.M., Remick, D.G., Ward, P.A. *et al.* Cellular and molecular regulation of tumour necrosis factor- α production by pentoxifylline. *Biochem Biophys Res Commun* 1988, **155**: 1230–1236.
8. Sullivan, G.W., Carper, H.T., Norick, W.J. & Mandell, G.L. Inhibition of the inflammatory action of interleukin and tumour necrosis factor (alpha) on neutrophil function by pentoxifylline. *Infect Immun* 1988, **56**: 1722–1729.
9. Tighe, D., Moss, R., Hynd, J. *et al.* Pentoxifylline reverses hemodynamic and histological changes associated with peritonitis in pigs. In: Mandell, G.L. & Novick, W.J. Jr (eds) *Pentoxifylline and Leukocyte Function*. HRP Inc., Somerville, NJ, 1988, pp. 184–189.
10. Ward, A. & Clissold, S.P. Pentoxifylline a review of its pharmacodynamic and pharmacokinetic properties, and its therapeutic efficacy. *Drugs* 1987, **34**: 50–97.
11. Bjorvatn, B., Bjertnaes, L., Fadnes, H.O. *et al.* Meningococcal septicemia treated with combined plasmapheresis and leucapheresis or with blood exchange. *Br Med J* 1984, **288**: 439–441.

asymptomatic arrhythmias can be induced by hypoglycaemia and may suggest a possible mechanism for the phenomenon of sudden death in insulin dependent diabetics.

M.A. Baxter
C. Garewal
R. Jordan
A.D. Wright
M. Nattrass

Department of Medicine,
General Hospital,
Birmingham B15 2TH, UK.

References

1. Collier, A., Matthew, D.M., Young, R.J. & Clarke, B.F. Transient atrial fibrillation precipitated by hypoglycaemia: Two case reports. *Postgrad Med J* 1987, **63**: 895–897.
2. Gale, E.A.M. Hypoglycemia and human insulin. *Lancet* 1989, **2**: 1264–1266.
3. Gale, E.A.M. The frequency of hypoglycemia in insulin-treated diabetic patients. In: Serrano-Rios, M., Lefebvre, P.J. (eds) *Diabetes 1985*. Elsevier, Amsterdam, 1986, pp. 943–947.
4. DCCT Research Group Diabetes Control and Complications Trials (DCCT), results of feasibility study. *Diabetes Care* 1987, **10**: 1–19.

Hypoglycaemia and atrial fibrillation

Sir,
We would like to report a further case of hypoglycaemia-induced self remitting atrial fibrillation. Although 2 similar cases have been previously reported in the *Journal*,¹ we feel that the recent reports of sudden death in young insulin dependent diabetics² may make it important to re-emphasize this interesting observation.

A 27 year old male with Type I diabetes of 15 years duration presented to the Accident and Emergency Department following a severe hypoglycaemic reaction. He was taking three daily injections of a Human Velosulin (6 units) and Insulatard (12 units) and claimed good glycaemic control which was supported by a serum fructosamine of 2.95 mmol/l (normal range 1.9–2.7) on admission. He had however been admitted to hospital on two previous occasions following severe nocturnal hypoglycaemia. There was no past medical or family history of cardiac arrhythmias.

At 20.00 h on the day of admission he became weak and confused. His mother recognized his symptoms and recorded a blood glucose of 1.4 mmol/l (BM 1–44 stick read by meter) before giving the patient milk. In the Accident and Emergency Department (21.00 h) he was given a further dose of oral glucose and a subsequent Reflocheck of 14 mmol/l was recorded. Although asymptomatic he was in atrial fibrillation with ventricular rate 100–150 beats/min, confirmed on electrocardiograph. Physical examination was normal and within one hour the rhythm spontaneously reverted to sinus. Subsequent cardiological assessment (auscultation, resting electrocardiogram and echocardiogram), serum potassium and thyroid stimulating hormone levels were all normal.

Hypoglycaemia is common in insulin treated diabetics^{3,4} but the incidence of associated cardiac arrhythmias remains unknown. This case supports the view that

Inappropriate secretion of vasopressin, hypopituitarism and corticosteroid therapy

Sir,
A recent report describes five patients with hypopituitarism and severe hyponatraemia due to the syndrome of inappropriate antidiuretic hormone secretion (SIADH) that improved within a few days after the institution of hydrocortisone therapy, whereas the infusion of normotonic or hypertonic saline had been found to be less effective.¹

We have observed the association between hyponatraemia due to SIADH and hypopituitarism in a 60 year old previously healthy woman who presented with lethargy, weakness and complete absence of axillary and pubic hair. Laboratory features revealed severe hyponatraemia (111 mmol/l), low plasma/urine osmolality ratio (230/298 mosmol/kg) and an increased urinary sodium excretion (50 mmol/l). Chest X-ray, serum creatinine and arterial blood gases were normal. She was treated by restricting fluid intake and with intravenous administration of 5% saline solution that restored serum sodium concentration and plasma osmolality within the first week of admission. A clinical diagnosis of panhypopituitarism was confirmed by means of the LHRH-TRH test as well as both acute and continuous ACTH stimulation performed after a stay of 7 days, at which time hormonal replacement was instituted.

Oelkers emphasize that a response to corticosteroids should be the hallmark of SIADH secondary to hypopituitarism. Although hydrocortisone can restore sodium excretion to normal by inhibiting vasopressin directly³ there is also evidence that it has mineralocorticoid-like properties and produces renal tubular sodium reabsorption in the absence of ADH.⁴ On the other hand, we re-established normal serum sodium levels after conventional therapy without corticosteroids. Probably plasma osmolality improvement itself after saline infusion may

cause inhibition of ADH secretion and maintenance therapy with hydrocortisone perpetuates this effect. Adequate treatment directed toward the underlying cause of SIADH is the most appropriate, but strict limitation of fluid intake will correct all the physiological disturbances despite persistence of the source of excessive antidiuretic activity.²

We suggest that hypopituitarism should be considered as a possible cause of unexplained hyponatraemia¹ regardless of its response to corticosteroid therapy.

J.L. Rodríguez-García
G. Fraile
V. Martínez

Service of Internal Medicine, Ramón y Cajal Hospital,
University of Alcalá de Henares, Madrid, Spain.

References

1. Oelkers, W. Hyponatremia and inappropriate secretion of vasopressin (antidiuretic hormone) in patients with hypopituitarism. *N Engl J Med* 1989, **321**: 492–496.
2. Kleeman, C.R. & Berl, T. The neurohypophyseal hormones: vasopressin. In: De Groot, L.J. *et al.* (eds) *Endocrinology*. Grune and Stratton, New York, 1979, pp. 253–270.
3. Ahmed, A.B., George, B.C., González-Auvert, C. & Dingman, J.F. Increased plasma arginine vasopressin in clinical adrenocortical insufficiency and its inhibition by glucocorticoids. *J Clin Invest* 1967, **46**: 111–123.
4. Axelrod, L. Glucocorticoid therapy. *Medicine* 1976, **55**: 39–48.

Phaeochromocytoma presenting with acute intestinal ischaemia and shock

Sir,

We have read with interest the case report of Carr *et al.* concerning small intestinal ischaemia occurring as a result of phaeochromocytoma.¹ We have recently had a similar experience with a patient who had a phaeochromocytoma in whom the presenting diagnosis was large bowel ischaemia with circulatory failure.

A 60 year old woman presented with a 4-hour history of colicky right hypochondrial pain and vomiting. She had noticed palpitations earlier that day; this symptom and mild hypertension had required β -blocker therapy in the past. Her only other medication was glibenclamide for the treatment of diabetes mellitus diagnosed 2 years previously.

On examination she was afebrile, oliguric, the systolic blood pressure was 80 mmHg and there was intense peripheral vasoconstriction. She had Kussmaul respiration and was tender in the right subcostal region; bowel sounds were sparse. Arterial blood gases showed metabolic acidosis. Abdominal X-ray films were normal. Bowel ischaemia was suspected and, whilst awaiting surgery, mesenteric angiography was performed. This showed moderate aortic atheroma but patency of the coeliac and both mesenteric axes.

At laparotomy after intravenous fluids the ascending colon was found to be ischaemic and this was treated by right hemicolectomy with terminal ileostomy and mucus fistula formation. Anaesthesia was complicated by hypertensive surges but the implication of this was not appreciated initially. Pathological examination of the surgical specimen confirmed ischaemic necrosis but there was no evidence of large artery occlusion although small fibrin

thrombi were seen in superficial sub-mucosal vessels. Post-operatively the patient required arterio-venous haemofiltration and ventilation on the ITU. Management was complicated by marked lability of blood pressure and the diagnosis of phaeochromocytoma was confirmed by assay of plasma catecholamines. Pulmonary gas exchange continued to deteriorate and the patient died 2 days later. At post-mortem a 10 cm diameter right adrenal phaeochromocytoma was identified which weighed 225 g.

Our patient presented with an acute abdomen and shock, and intestinal ischaemia was diagnosed at laparotomy. We suspect that the intestinal pathology resulted from catecholamine-induced vascular spasm. Indeed, severe vasoconstriction which has been found to be responsible for pulseless limbs and peripheral gangrene as well as permanent neurological deficits such as cortical blindness, is a well-recognized accompaniment of phaeochromocytoma.² Such intense vasoconstriction may have contributed to the acute renal failure that developed in this case, although other factors such as sepsis and shock were possibly more important. An alternative explanation of the intestinal ischaemia is a 'watershed-type' infarction as a consequence of hypotension (perhaps induced by tumour necrosis) in a patient with pre-existent atheromatous disease. Infarction of a phaeochromocytoma is well-recognized³ and its presentation of shock and acute abdominal pain has resulted in several unexpected fatalities during the anaesthesia and surgery of an 'acute abdomen'.⁴

Although we do not feel that the outcome would have been altered in this case we wish to re-emphasize the need to consider a diagnosis of phaeochromocytoma in patients with acute abdominal signs whose presentation is atypical, and we would certainly support the view of other authors^{1,5} that gastrointestinal symptoms occurring in association with phaeochromocytoma should indicate the necessity for urgent tumour removal, as gut ischaemia may be imminent.

P.A. Kalra
E.W. Thornton-Chan
C. Weinkove¹
B. Bowles²
G. Ingram³
S. Waldek
A.M. Holmes

Renal Unit and Departments of

¹Clinical Biochemistry, ²Intensive Care and

³Surgery, Hope Hospital, Eccles Old Road,
Salford, UK.

References

1. Carr, N.D., Hulme, A., Sheron, N., Lees, W.R. & Russell, R.C.G. Intestinal ischaemia associated with phaeochromocytoma. *Postgrad Med J* 1989, **65**: 594–596.
2. Radtke, W.E., Kazmier, F.J., Rutherford, B.D. & Sheps, S.G. Cardiovascular complications of phaeochromocytoma crisis. *Am J Cardiol* 1975, **35**: 701–705.
3. Bergland, B.E. Phaeochromocytoma presenting as shock. *Am J Emerg Med* 1989, **7**: 44–48.
4. Ledingham, J.G.G. Secondary hypertension-Phaeochromocytoma. In: *Oxford Textbook of Medicine*, 2nd Ed., vol. 2. 13, 1988, pp. 392–396.
5. Fee, H.J., Fonkalsrud, E.W., Ament, M.E. & Bergstein, J. Enterocolitis with peritonitis in a child with phaeochromocytoma. *Ann Surg* 1977, **185**: 448–450.